A successful renal transplantation in Behçet’s syndrome

Renal involvement is not frequent in Behçet’s syndrome (BS) and consists of occasional reports of patients suffering from glomerulonephritis, IgA nephropathy and renal amyloidosis. We present the successful outcome of a renal transplantation in a patient who had end stage renal failure secondary to glomerulonephritis. To our knowledge, this is the first patient with BS to receive an organ transplantation.

The detailed history of this patient at the time of the diagnosis of glomerulonephritis was the subject of a case report in 1991. In brief, she was 21 years old when she developed recurrent oral and genital ulcers, bilateral uveitis, erythema nodosum, folliculitis, and intermittent arthritis of the knee. Two years later, she was referred to our centre for further evaluation of eye symptoms. She had no active mucocutaneous lesions at that time, the pauchy reaction was positive and she carried HLA BS. It was decided to prescribe only local drops for her mild eye involvement. Three months later she experienced two ocular episodes resulting in a sharp decline of visual acuity and azathioprine 2.5 mg/kg/day was prescribed. Two weeks later she was admitted to the hospital because of microscopic haematuria. She was ANA negative, the anti-DNA and serum complement levels were within normal range. Her glomerular filtration rate was 67 ml/min. An open renal biopsy showed diffuse proliferative glomerulonephritis and weak focal segmental positivity of IgA and IgM. She was treated with three boluses of 1 g methylprednisolone and was discharged prescribed azathioprine 150 mg/day, aspirin 300 mg/day and prednisone 30 mg/day. She was well except for occasional mucocutaneous symptoms and a mild local ocular episode during the next four years. However her renal function deteriorated progressively despite uninterrupted treatment with azathioprine and changing doses of prednisone and she was put on regular haemodialysis in August 1994. In the 14th month of haemodialysis, she received a kidney from her mother. The graft function started immediately and she was prescribed maintenance immunosuppression with azathioprine, cyclosporin A and methylprednisolone. An acute interstitial type rejection on the 11th day of transplantation was treated successfully with pulsed corticosteroids. Now 40 months after transplantation, she has normal renal function and is free of any symptoms of BS except for occasional oral ulcers.

We had some hesitation in performing a renal transplantation on our patient initially because of the lack of any previous experience and particularly because of our concern for the heightened inflammatory response of BS patients to simple penetrating trauma that is best characterised by the pauchy reaction. This reaction, however, is not only limited to the skin and development of aneurysms after vascular punctures and episodes of synovitis after arthrocentesis have been observed. Furthermore, postoperative complications leading to a poor outcome such as occlusions of grafts/anastomoses after the surgical treatment of aneurysms or perivalvular leakage and suture breakdown after aortic valve replacement have been reported in BS patients. As these complications are probably related to the pathergy phenomenon of BS, you would also reasonably expect problems after an organ transplantation, an operation with arterial and venous anastomoses. On the other hand, we had previously shown that despite the increased inflammation, wound healing after full thickness skin punch biopsies is not changed in BS. We have not experienced any of the feared complications after the transplantation procedure in this instance. Our concern for this favourable outcome might be that our patient was female. It is known that BS runs a milder disease course in women compared with men. Additionally, the rather intensive immunosuppressive/anti-inflammatory post-transplant drug use might also have contributed to the diminished disease activity of our patient as well as to the prevention of a reaction at the site of the donor kidney. Whatever it might be related to, the outcome in our patient suggests that BS patients can undergo renal transplantation with a satisfactory outcome.

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Lymphocyte phenotypes in systemic sclerosis

Although the pathophysiology of systemic sclerosis (SSc) is not fully clarified, there are considerable data implicating abnormalities of microvascular changes, fibroblast activation and immune system abnormalities. Immune system activation may be induced as a stimulus in both fibrotic and vascular damage. To investigate the immune system abnormalities in the pathogenesis of SSc we evaluated lymphocyte phenotypes in patients with SSc and healthy controls. We used the cytokine profile (Epics Profile II) for total T (CD3), T helper (CD4), T suppressor (CD8), B lymphocyte cell surface marker (CD19), activation marker (CD25) and natural killer (NK) cell surface marker NKH-1 (CD56).

We studied 29 patients (27 women, two men) 16 limited, 12 diffuse and one overlap who fulfilled preliminary criteria for classification of SSc. Anti-nuclear antibody was positive in 25 (86.2%) and anti-Sc170 antibodies was positive in seven (24.1%) patients. The age range of the patients was 20–63 years (mean (SEM) 40 (5)) and the mean (SEM) disease duration was 5.6 (5.5) years. Patients were receiving no medication nor had received any immunosuppressive agent for at least three months. Controls were 12 aged sex matched healthy volunteers with an age range from 27–51 years. Data were compared for significance for Student’s unpaired t test.

Table 1 summarises lymphocyte phenotypes in patients with SSc and healthy controls.

Table 1 summarises lymphocyte phenotypes in patients with SSc and healthy controls.

<table>
<thead>
<tr>
<th>Serum</th>
<th>Systemic sclerosis (n=29)</th>
<th>Healthy controls (n=12)</th>
<th>t Test</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>CD3 (%)</td>
<td>71 (9)</td>
<td>69 (9)</td>
<td>0.660</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>CD4 (%)</td>
<td>44 (9)</td>
<td>45 (9)</td>
<td>0.110</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>CD8 (%)</td>
<td>31 (9)</td>
<td>25 (9)</td>
<td>1.914</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>CD4/CD8</td>
<td>1.56 (0.6)</td>
<td>1.84 (0.6)</td>
<td>1.339</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>CD19 (%)</td>
<td>12 (4)</td>
<td>13 (5)</td>
<td>0.445</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>CD25 (%)</td>
<td>18 (9)</td>
<td>7.1 (3)</td>
<td>4.150</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>CD56 (%)</td>
<td>22 (9)</td>
<td>14 (5)</td>
<td>2.691</td>
<td>&gt;0.05</td>
</tr>
</tbody>
</table>

*Unpaired Student’s t test. Data shown as mean (SD).
various results in the pathogenesis of SSc, of CD56+ cells in the pathogenesis of SSc, and of CD8+ and CD56+ T lymphocytes is still obscure. Presence of autoantibodies and hypergammaglobulinemia support the role of humoral immunity but B lymphocytes were rarely found in the skin biopsy specimens. CD19+ is a cell surface marker of B lymphocytes and we could not observe any difference in the levels of CD19+ and CD16+ NK cells in SSc group, our findings can say that B lymphocytes might play only a minor part in the pathogenesis of SSc. CD25+ is one of the subunits of high affinity IL2R and known as the alpha chain of IL2R. Bruns et al established a clear correlation between CD25+ and soluble IL2R in serum. T lymphocytes expressing CD25+ and T helper cell derived cytokines and growth factors stimulate matrix protein synthesis by fibroblasts, resulting in generalised fibrosis and sclerosis. In our study we found significant increase of CD25+ and this surface marker can be used in the follow up the inflammatory stage and activity of SSc. In further studies the investigation of CD25+ T cell subsets CD4+, CD8+, TCR gamma-delta and other T cell activation markers HLA-DR, CD45RO/CD45RA will be useful to shed light on the pathogenesis of SSc. NK cell abnormalities have been described in a number of rheumatic diseases such as RA, Sjogren’s syndrome, systemic lupus erythematosus. NK cells are large granular lymphocytes easily identified morphologically by the presence of azurophil granules in their cytoplasm and they commonly express the presence of azurophil granules in their cytoplasm and they commonly express CD16+ and CD56+, CD56+ is a homofolic acid molecule that belongs to the immunoglobulin superfamily. NK cells are the main cellular body production, but in SLE, B cells have increased reactivity to this cytokine. Therefore, the decreased pericardial lymphocyte infiltration and the increased reactivity to this cytokine, as well as the increased cytokine concentration in pericardial fluid, might indicate an immune response to this cytokine.

**Lymphocyte populations and cytokine concentrations in pericardial fluid from a systemic lupus erythematosus patient with cardiac tamponade**

Pericardial involvement is the most common cardiovascular complication in systemic lupus erythematosus (SLE). The clinical picture varies from subclinical pericardial effusion and classic acute pericarditis to cardiac tamponade. Immunological studies of pericardial fluid (PF) have been limited to determination of autoantibodies, complements and immune complexes. To further study the pathogenic mechanisms involved in lupus pericarditis, we determined the lymphocytic populations and cytokine concentration pattern in PF and peripheral blood (PB) from a SLE patient with cardiac tamponade.

We report a case of a 38 year old man with SLE diagnosed in December 1995 when he presented with polyarthritis, photosensitivity, oral ulcers, nephritis, non-hemolytic anemia, positive ANA, high titer of anti-dsDNA and hypocomplementemia. The patient improved with corticosteroid and intravenous cyclophosphamide treatment. However, on 18th July 1997 he presented with syncope, hypotension (80/40 mm Hg), a tachycardia, jugular vein distension and cardiomegaly. The two dimensional echocardiogram showed a large pericardial effusion with right atria and ventricle collapse in diastole. Pericardioscintigraphy was performed and 180 ml of an orange fluid was aspirated. Examination of PF showed white blood cell count of 5280/mm³ (polymorphonuclear cells = 96%). The absolute number of lymphocytes was lower in PF than in PB (211 e 700/mm³). PF

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Peripheral Blood</th>
<th>Pericardial Fluid</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lymphocyte population (%)</td>
<td>57.8</td>
<td>49.0</td>
</tr>
<tr>
<td>CD4+ T cells</td>
<td>17.6</td>
<td>25.0</td>
</tr>
<tr>
<td>CD8+ T cells</td>
<td>34.3</td>
<td>25.0</td>
</tr>
<tr>
<td>B cells</td>
<td>7.8</td>
<td>8.3</td>
</tr>
<tr>
<td>NK cells</td>
<td>34.3</td>
<td>41.7</td>
</tr>
<tr>
<td>Cytokine concentration (pg/mL)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IL1β</td>
<td>3.0</td>
<td>2400</td>
</tr>
<tr>
<td>IL6</td>
<td>24.0</td>
<td>&lt;4.0</td>
</tr>
<tr>
<td>IL4</td>
<td>&lt;6.0</td>
<td>&lt;6.0</td>
</tr>
<tr>
<td>IL10</td>
<td>&lt;3.5</td>
<td>139.8</td>
</tr>
<tr>
<td>TNFα</td>
<td>3.8</td>
<td>15.4</td>
</tr>
<tr>
<td>INFγ</td>
<td>1.5</td>
<td>32.8</td>
</tr>
</tbody>
</table>

*Manufacturer (Genzyme, Boston, MA) detection limits: 3 pg/ml for IL1β, INFγ, TNFα and TNFβ; 4 pg/ml for IL2; 6 pg/ml for IL6; 18 pg/ml for IL8; and 5 pg/ml for IL10.

The level of protein was 4.1 g/dl (serum = 5.3 g/dl), glucose was 53 mg/dl (serum = 110 mg/dl) and LDH was 471 IU/l (serum = 110 IU/l). PF cultures were negative. No malignant cells were seen to suggest malignancies. The elevated SLE disease activity index (SLEDAI) was due to high dose corticosteroids and azathioprine. Prednisone was gradually decreased to 10 mg daily over a three month period. After a 22 month follow up, he remained clinically stable without recurrent pericardial involvement or SLE exacerbations.

Before starting immunosuppressive treatment, PF and PB were obtained simultaneously for immunological analysis. Mononuclear cells from both sources were isolated by gradient centrifugation and the frequency of lymphocyte populations was determined by flow cytometry. The cytokine concentrations from plasma and PF were determined by ELISA. Table 1 shows the results. Among lymphocytes, the percentage of CD4+ T cells and NK cells was higher in PF, while the frequency of CD8+ T cells was higher in PB. IL6 concentration was much higher in PF than plasma. Also, IL1β and IL10 concentrations were higher in PF. IL2 was detected in plasma but not in PF.

The considerable increase in pericardial IL6, with respect to plasma or PB, was of particular interest. PF concentrations of IL6 in our patient were substantially higher than those observed in PF from patients with inflammatory and non-inflammatory heart conditions. However, we did not find any correlation of increased reactivity to this cytokine, as well as the increased cytokine concentration in pericardial fluid, might indicate an immune response to this cytokine. As in our case, IL6 is usually expressed or increased in the affected organ or system rather than in plasma. IL6 has been found to be higher in cerebrospinal fluid and urine than in serum of SLE patients with CNS disease and active nephritis respectively.

The decreased pericardial lymphocyte count and fluid characteristics observed here are in agreement with other studies. The higher frequency of CD4+ T cells and NK cells in PF could be associated with the observed cytokine concentration pattern. For example, CD4+ memory T cells from SLE patients highly secrete IL10 compared with normal controls.

In summary, different patterns of lymphocyte populations and cytokines were found in both sources, with type 2 cytokines predominating in PF and type 1 in PB. Further studies would be required to confirm these results presented here. In addition, immunocytochemical studies of pericardial

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**Table 1 Frequency of lymphocyte populations and cytokine concentrations in peripheral blood and pericardial fluid**

Parameter | Peripheral Blood | Pericardial Fluid |
<table>
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In summary, different patterns of lymphocyte populations and cytokines were found in both sources, with type 2 cytokines predominating in PF and type 1 in PB. Further studies would be required to confirm these results presented here. In addition, immunocytochemical studies of pericardial
Anterior uveitis (AU) is the most common and immunological lymphocyte and cytokine profiles may differ between pericardial fluid and tissue.

Table 1 Lymphocyte populations in AU patients and controls

<table>
<thead>
<tr>
<th>AU patients (n=146)</th>
<th>Controls (n=31)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lymphocytes (no/mm³)</td>
<td>2425.60 (964.44)</td>
<td>2567.74 (820.72)</td>
</tr>
<tr>
<td>CD3 (no/mm³)</td>
<td>1734.20 (726.67)</td>
<td>1835.64 (586.68)</td>
</tr>
<tr>
<td>CD4 (no/mm³)</td>
<td>1023.91 (489.16)</td>
<td>1719.7 (787.76)</td>
</tr>
<tr>
<td>CD8 (no/mm³)</td>
<td>42.56 (9.50)</td>
<td>47.00 (6.13)</td>
</tr>
<tr>
<td>CD4/CD8</td>
<td>71.96 (8.20)</td>
<td>71.27 (4.28)</td>
</tr>
<tr>
<td>CD19 (no/mm³)</td>
<td>29.25 (8.81)</td>
<td>26.72 (6.81)</td>
</tr>
<tr>
<td>CD4CD45R+ (no/mm³)</td>
<td>1.70 (0.89)</td>
<td>1.02 (0.78)</td>
</tr>
<tr>
<td>CD4CD45R− (no/mm³)</td>
<td>226.87 (227.44)</td>
<td>335.90 (142.35)</td>
</tr>
<tr>
<td>CD4 (%)</td>
<td>10.81 (5.65)</td>
<td>13.64 (5.09)</td>
</tr>
<tr>
<td>CD8 (%)</td>
<td>71.27 (8.20)</td>
<td>71.27 (4.28)</td>
</tr>
<tr>
<td>CD4CD45R+ (%)</td>
<td>16.70 (9.99)</td>
<td>25.20 (7.76)</td>
</tr>
<tr>
<td>CD4CD45R− (%)</td>
<td>50.63 (13.84)</td>
<td>55.49 (12.54)</td>
</tr>
<tr>
<td>CD8 (%)</td>
<td>16.70 (9.99)</td>
<td>25.20 (7.76)</td>
</tr>
<tr>
<td>CD4CD45R+ (%)</td>
<td>21.90 (17.96)</td>
<td>25.20 (7.76)</td>
</tr>
<tr>
<td>CD4CD45R− (%)</td>
<td>25.20 (7.76)</td>
<td>28.50 (6.40)</td>
</tr>
</tbody>
</table>

AU = anterior uveitis, SA = spondyloarthritis.

**Figure 1** Absolute values of CD4CD45R+ cells. Patients with low values had lower percentages of CD4CD45R+ cells than controls, and percentages lower than those of SA patients (p<0.001). IAU = idiopathic anterior uveitis; AU = anterior uveitis; SA = spondyloarthritis.

**Figure 2** Percentages of CD4CD45R− cells. Patients with AU had higher percentages than the healthy subjects and SA patients (p<0.001). Abbreviations as in figure 1.
we are indebted to Ms E Velasco for assistance in the preparation of the manuscript.

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4 Brewerton DA, Cañellas, Josep Pujol, Anna Lafont. Roselló, Javier Arasa, Marta Larrosa, Genonima Pons, Isabel Rotés, Raimon Sanmartí, Eduardo Germans Trias i Pujol, C/ de Canyet s/n, 08006 Barcelona, Spain.


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We appreciate the comment by Olivel et al on our article on RS3PE. They reviewed 27 previously described RS3PE patients after a follow-up of six years.

As we suggested in a previous report, we confirmed that RS3PE syndrome should be considered a heterogeneous condition associated with different inflammatory rheumatic diseases and also with neoplastic disorders.

In our study none of the 23 patients with RS3PE syndrome developed clinical features supporting the diagnosis for another disease. The different study design and selection of patients may in part explain the subset of patients with other diseases and with a worse prognosis observed by Olivel et al.

We designed a prospective follow up study excluding patients satisfying the criteria for the diagnosis of polyalgia rheumatica, rheumatoid arthritis and seronegative spondyloarthopathies. Moreover, patients with a clinical history of cancer were excluded from the study. In their original report these authors performed a retrospective study including all patients with remitting distal extremity swelling with pitting oedema. They recruited also patients not evaluated for spondylarthropathies, which may be associated with distal extremity swelling with pitting oedema.

However, in their retrospective evaluation Olivel et al found that 13 of 22 (59%) patients were asymptomatic and drug free over a six year follow up period, confirming that RS3PE not associated with other conditions and with a good prognosis does exist.

The problem is how to label this clinical picture. As discussed in our article, the similarities of demographic, clinical and MRI findings between patients with “pure” RS3PE syndrome and those with polyalgia rheumatica and the concurrence of the two syndromes suggest that these conditions may be part of the clinical spectrum of the same disease. In the series of Olivel et al the patient with a clinical course characterised by alternate relapses of HLA B27 positive seronegative oedema or polyalgic symptoms further supports our hypothesis. Even those RS3PE patients successively diagnosed as having seronegative rheumatoid arthritis (elderly onset rheumatoid arthritis) do not conflict with our conclusions. Healey described patients who developed episodes of polyalgia rheumatica and seronegative rheumatoid arthritis at different times during follow up.

Similar clinical characteristics have been recently described in a population based cohort of patients with giant cell arteritis followed up over a 42 year period. Four of the six patients who fulfilled the criteria for the diagnosis of rheumatoid arthritis during the follow up experienced multiple separate episodes of symmetrical arthritis, proximal symptoms of polyalgia rheumatica and distal extremity swelling with pitting oedema.

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RS3PE: six years later

We read with interest the paper by Cantini et al and would like to comment on it.

In 1992 we performed a retrospective multicentre study of 27 RS3PE patients. We concluded that personal history of polyalgia rheumatica (two patients), presence of erosions (one patient) and evolution to haematological diseases (two patients concomitantly developed a T lymphocytopenia in one and a myelodysplastic syndrome) suggested that RS3PE syndrome might not be a distinct clinical entity. At that moment 12 patients were asymptomatic and 12 required treatment. This was reported elsewhere.

Now, six years later, we have reviewed the original cohort of patients with the RS3PE syndrome. A questionnaire was sent to the participating rheumatologists. The survey focused on articular symptoms, treatment and evolution. The current cohort was composed of 22 patients (male 16; female 6; mean age:77.9; range 64–91). Four patients died (the three with haematological diseases, one stroke) and one was not located. Thirteen patients were asymptomatic and without treatment, in contrast none required treatment, namely corticosteroids (6), gold salts (1), chloroquine (1) and NSAID (1). Interestingly, two of the patients were identified by their rheumatologist as having a seronegative rheumatoid arthritis, another patient had a chronic disease with separate corticosteroid responsive episodes of bilateral hand oedema and polyalgic symptoms at different times. Last but not one patient developed Raynaud’s phenomena, both hands had sclerodactyly. A nailfold capillary microscopy showed a decreased number of capillary loops, which were widened, suggesting systemic sclerosis.

Our results suggest that RS3PE syndrome has a good prognosis. More than half of the patients are asymptomatic and without treatment six years later. However, there is a subset of patients that have other diseases. Although pure RS3PE syndrome does exist the evolution should be carefully monitored.

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Letters, Matters arising
Correspondence to: Dr F. Cantini, 2nd Divisione di Medicina, Ospedale di Prato, Piazza Ospedale 1, 59100 Prato, Italy.

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10 McCarthy GM, Kurup PV, Westfall PR. Basic 

11 McCarthy GM, Kurup PV, Westfall PR. Basic 

12 McCarthy GM, Kurup PV, Westfall PR. Basic 

13 McCarthy GM, Kurup PV, Westfall PR. Basic 

14 McCarthy GM, Kurup PV, Westfall PR. Basic 

15 McCarthy GM, Kurup PV, Westfall PR. Basic 

16 McCarthy GM, Kurup PV, Westfall PR. Basic 

17 McCarthy GM, Kurup PV, Westfall PR. Basic 

18 McCarthy GM, Kurup PV, Westfall PR. Basic 

19 McCarthy GM, Kurup PV, Westfall PR. Basic 

20 McCarthy GM, Kurup PV, Westfall PR. Basic 

21 McCarthy GM, Kurup PV, Westfall PR. Basic 

22 McCarthy GM, Kurup PV, Westfall PR. Basic 

23 McCarthy GM, Kurup PV, Westfall PR. Basic 

24 McCarthy GM, Kurup PV, Westfall PR. Basic 

25 McCarthy GM, Kurup PV, Westfall PR. Basic 

26 McCarthy GM, Kurup PV, Westfall PR. Basic 

27 McCarthy GM, Kurup PV, Westfall PR. Basic 

28 McCarthy GM, Kurup PV, Westfall PR. Basic 

29 McCarthy GM, Kurup PV, Westfall PR. Basic 
tion, at 3.4 per 1000 per year at age 51 and 9.0 per 1000 per year at age 61 are presumably higher than those in long lived Malmöhus County, Sweden.

Of interest, in our own larger study with meticulously computed “expected” values in four different populations we also had an “expected” death rate of about 10% to 15% over 10 years. But, these were not inception cohorts and their age at start of follow up was 60.4, 62.6, 59.8, and 69.1 years. Thus, they were much older cohorts. Given the expected doubling of mortality rates each eight years (Gompertz’s law), expected deaths should have been two to three times more in our cohorts than in a cohort beginning at age 51.

Finally, recent studies have not suggested that “rheumatoid” deaths in themselves are the cause of the increased mortality in RA. The observed “excess” deaths are spread around in multiple disease categories, with accelerated atherosclerosis numerically the largest problem and only a slight relative increase in systemic RA complications, gastrointestinal haemorrhage, and infections.

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Authors’ reply

We were pleased to notice the interest in our paper shown by Drs Fries and Bloch. In reply to their comments we do not consider the death rate of 10% in the cohort as an excessive one compared with the age and sex matched general population. It is not possible to calculate more precise figures of expected deaths knowing the mean age of the cohort only. To clarify this and make comparison possible we enclose a table of the age distribution in our cohort in five year intervals giving the number of observed and expected deaths for each age interval separately.

Women do live longer in Malmöhus County, Sweden than in the US. Female mortality rates in Malmöhus County were 3.76 per 1000 at age 51 and 7.32 per 1000 at age 61 in 1985. In 1996 the corresponding figures were 2.03 per 1000 at age 51 and 3.39 per 1000 at age 61.

We agree that the main cause of death in RA patients very seldom is the rheumatoid disease in itself. This was true also for our study where no certain connection between RA and death was found in any of the cases.

Table 1

<table>
<thead>
<tr>
<th>Age group</th>
<th>Number of patients</th>
<th>Expected mortality</th>
<th>Observed mortality</th>
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<tbody>
<tr>
<td>15-19</td>
<td>1</td>
<td>0</td>
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</tr>
<tr>
<td>20-24</td>
<td>3</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>25-29</td>
<td>7</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>30-34</td>
<td>3</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>35-39</td>
<td>15</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>40-44</td>
<td>20</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>45-49</td>
<td>27</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>50-54</td>
<td>34</td>
<td>2</td>
<td>2</td>
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<tr>
<td>55-59</td>
<td>24</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>60-64</td>
<td>19</td>
<td>3</td>
<td>2</td>
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<td>65-69</td>
<td>16</td>
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<td>3</td>
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<tr>
<td>70-74</td>
<td>11</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>75-79</td>
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<td>2</td>
<td>2</td>
</tr>
<tr>
<td>All</td>
<td>183</td>
<td>20</td>
<td>18</td>
</tr>
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Crystals in arthritis: new age nonsense or novel therapeutic target?

GERALDINE M MCCARTHY

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